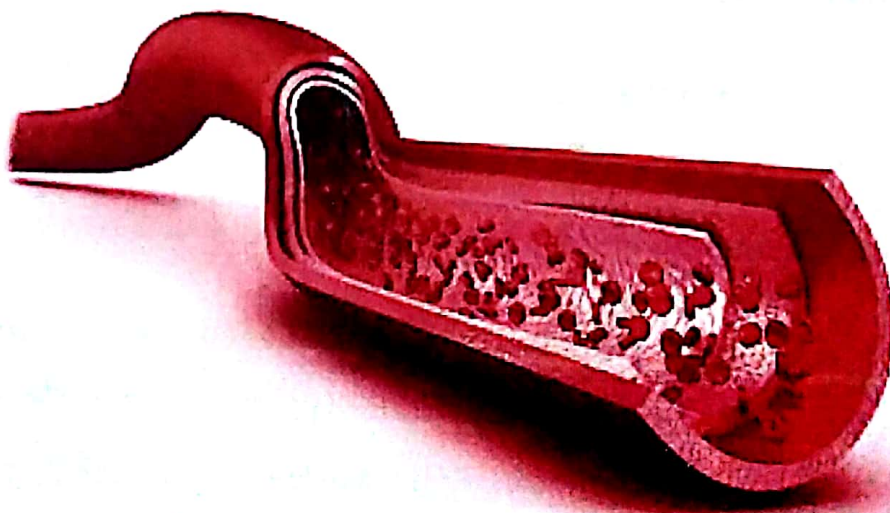


Easy Pathology

— Dr. Abdelrahman Khalifa —

Chapter 4

Hemodynamic Disorders



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Edema

Pathological Accumulation of fluid in interstitial tissue and body spaces
 Anasarca: generalized edema affecting S.C. tissue, serous sacs, and viscera
Factors affecting fluid accumulation:
 Increased hydrostatic pressure in capillaries – reduced osmotic pressure



Causes & pathogenesis:

Hydrostatic (increased hydrostatic pressure in veins and capillaries)

- **Generalized** : Right s. H.F. → generalized venous congestion → inc. H. Pressure
- **Localized**: Venous obstruction (Thrombus DVT) , Vein compression (Tumor, pregnancy)

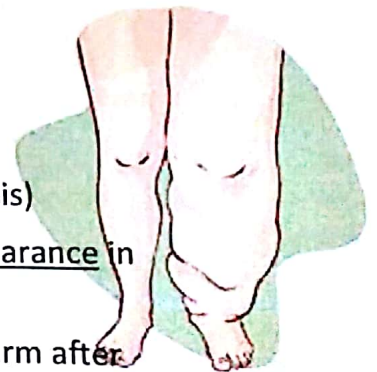
Osmotic (decreased plasma albumin → decreased osmotic pressure of plasma → edema)

- **Nutritional** (decreased intake – increased loss e.g. Famines & starvation)
- **Enteropathy with protein loss** (Maldigestion or malabsorption)
- **Liver failure** → decreased synthesis of albumin
- **Nephrotic syndrome** → proteinuria (> 3gm/day)

Hypoalbuminemia → decreased plasma osmotic pressure → edema → decreased renal perfusion → salt and water retention

Lymphatic (Lymphatic obstruction → lymph fluid accumulates in tissue)

- **Congenital aplasia** (Milroy disease)
- **Lymphangitis** e.g. Filariasis → Edema of L.L and scrotum (elephantiasis)
- **Malignant tumors** → lymphatic obstruction (e.g. Peau de orange appearance in breast cancer)
- **L.N. removal** (e.g. In mastectomy → L.N. removal → edema of the arm after few years)
- **Radiotherapy** → following cancer breast → edema of the arm



Na & water retention → increased hydrostatic P.

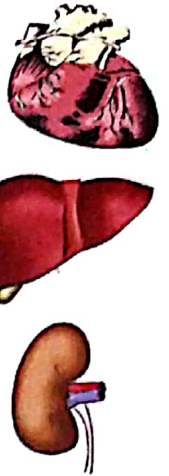
- **Nephritic syndrome** → inflammation of the kidney → Renin → increase aldosterone
- **Adrenal cortical tumors** → hyperaldosteronism → salt & water retention
- **Right sided H.F.** → renal hypoxia → Renin → aldosterone

Increased capillary permeability (inflammatory mediators , or hypoxia)

Types of edema

Generalized edema

- **Cardiac** (start in both L.L. → then generalized "anasarca")
 - Right S. H.F. → general venous congestion → increased hydrostatic pressure
 - H.F. → real hypoxia → Renin → aldosterone → salt & water retention
 - H.F. → capillary hypoxia → increased permeability
- **Hepatic** (start as Ascitis → then generalized)
 - Cirrhosis → portal hypertension → ascitis
 - Liver failure → HypoAlbuminemia → osmotic edema
- **Renal** (start periorbital → then generalized)
 - Nephritic (G.N. → Renin → aldosterone)
 - Nephrotic (Proteinuria → decreased osmotic pressure)
- **Neutritional** (hypoproteinemia) starts periorbital

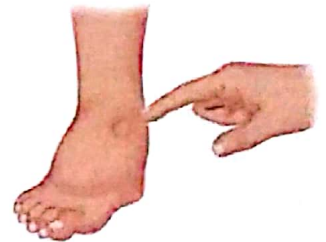


Localized edema

- **Obstructive** (venous obstruction – lymphatic obstruction)
- **Inflammatory**

Pathological features

- **Subcutaneous edema**
 - **Pitting** (soft)
 - **Non pitting** (Hard): lymphatic edema → excess C.T. proliferation → firm
- **Sites:** Cardiac edema starts in L.L. and scrotum, renal edema is periorbital
- **Effusion** (fluid in serous sacs, e.g. hydrothorax, pericardial effusion)
- **Pulmonary edema:** due to left sided heart failure, Mitral stenosis, or due to ARDS → Lung congestion → increased hydrostatic pressure in pulmonary capillaries → accumulation of transudate in alveoli.
 - Lung is heavy – C/S is frothy & bloody
- **Microscopic picture & lab analysis:**
 - Separation of EC matrix
 - **Exudate** : protein >4gm, S.G.>1020 **Transudate** protein <1gm%, S.G.<1012



Complications of edema

Skin: cellulitis & delayed wound healing

Lung: Infection, decreased ventilation

Cerebral: Compression & herniation

Congestion

Passive Accumulation of blood inside veins, due to obstruction or decreased venous return

Congestion	Hyperemia
Passive <u>venous</u> obstruction	Active <u>arteriolar</u> dilatation
Decreased blood flow	Increased blood flow
Veins and capillaries	arterioles
Pathological (generalized – localized)	-Physiological (digestion or muscle exercise) -Pathological (inflammation)
deoxygenated blood → Tissue hypoxia	Increased Oxygen supply



Causes of Congestion

- Localized
 - Venous thrombosis (e.g. DVT)
 - Compression (e.g. Tumors, portal fibrosis, Pregnancy)
 - Ligation or twisting (e.g. surgical ligation, intestinal volvulus)
- Generalized
 - **Acute generalized venous congestion :**
Acute H.F. (e.g. *Pulmonary embolism*)
 - **Chronic generalized venous congestion:** Chronic Rt. sided. heart failure
caused by: *Mitral stenosis – Pulmonary stenosis – Lung fibrosis - Emphysema*

Pathological manifestations of Chronic generalized venous congestion

1. Cyanosis (due to hypoxia)
2. Dyspnea
3. Congested pulsating neck veins
4. Bilateral lower limb edema → then generalized edema (mechanisms of edema?)
5. Organ Congestion: (e.g. Liver congestion → large tender)

Liver congestion (Nut-meg)

Gross: Large, *Nutmeg color* (brown hemosiderin + yellow fatty degeneration)

Microscopic:

- C.V. & sinusoids: dilated congested
- Cells: necrosis in central zone, fatty change in midzone
- Kupffer cell contain hemosiderin

Fate: Hemosiderosis → Cardiac cirrhosis



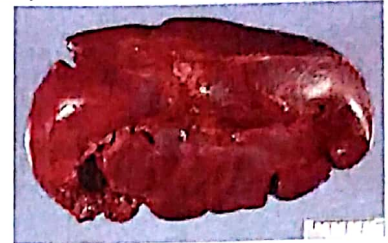
Splenic congestion

Gross: Large, dark red

Microscopic:

- **Red pulp & sinusoids:** dilated congested → ruptured → hemorrhage → forming **gandy-gamna nodules** (hemosedrin + Fibrosis)
- **White pulp:** Atrophic lymphoid follicles
- **Littoral cell** contain hemosedrin

Fate: Hemosidrosis → fibrosis & **Hypersplenism**



Lung congestion

(N.B. Lung is congested in case of Mitral stenosis, or Left sided heart failure)

Gross: Large, dark red (bloody & frothy C/S) → brown & firm (**brown induration**)

Microscopic:

- **Capillaries:** dilated & congested → ruptured capillaries
- **Veins:** fibrosis of intima – hypertrophy of media
- **Alveoli:** Thick wall (edema) - lumen contain RBCs & transudate.
- **Macrophage** in the wall of alveoli → engulf hemosedrin → heart failure cells
- **Arterioles:** (elastic hyperplasia & hyalinosis) in case of pulm. hypertension

Fate: hemosidrosis → lung Fibrosis (**brown induration**)

Clinical presentation: Hemoptysis



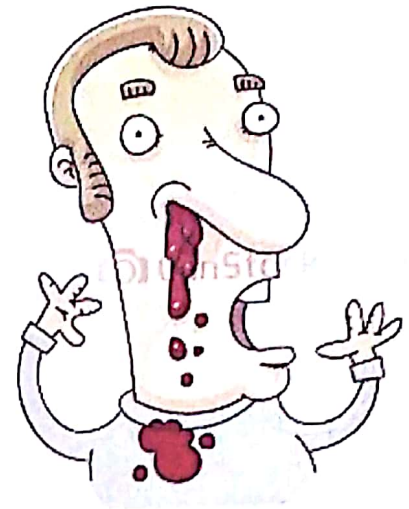
Hemorrhage

Escape of blood outside CVS.

Causes:

- **Traumatic** : Direct injury of vessels
- **Spontaneous** :
 - **General**
 - Hypertension
 - Bleeding tendency (pupura – hemophilia)
 - Leukmia
 - Vit. C, K deficiency

Bleeding comes from capillaries
 - **Local**
 - Vascular damage:
 - Arterial: Aneurism – Atherosclerosis - Arteritis
 - Venous: Varicose veins
 - Capillaries: Congestion
 - Vascular penetration: Tumors – TB – perforated ulcer



Types of hemorrhage:

- **External** (eg. Epistaxis, hematemesis, hemoptysis, hematuria, melena "blood from upper G.I.T", bleeding per rectum "blood from lower G.I.T.")
- **Hmatoma** deep internal hemorrhage
- **Subcutaneous** :
 - Petechiae: pinpoint spots in subcutaneous tissue and mucous membranes
 - Purpura > 3 mm , more superficial
 - Ecchymosis > 1cm

Subcutaneous hemorrhage begins **Red or Blue** → then **brown** (hsdrin) → then yellow and disappear by macrophage

Effects & complications of hemorrhage:

- **Small amount**: < 20% → no complications
- **Large amount**: >20% (1 L.) → Hypovolemic shock
- **Repeated small amounts or Chronic loss**: Iron deficiency anemia
- **Localized hemorrhage** → Hemosidrosis & organ fibrosis

N.B. natural haemostasis is done by: Local vasoconstriction , Clot, then amount is compensated by Tachycardia, bone marrow hyperplasia, & plasma protein restoration.

Shock

Generalized hypoperfusion to vital organs

Cardiogenic	Hypovolemic	Septic	Neurogenic	Anaphylactic
-Myocardial diseases -Pulmonary embolism	- <u>Dehydration</u> : vomiting, diarrhea, burns - <u>Hemorrhage</u>	- <u>Septicemia</u> (G -ve enotoxin) → high levels of LPS & cytokines	- <u>Anesthesia</u> - <u>Nerve injury</u> <i>cause 1- severe pain 2- motion</i>	- <u>Hypersensitivity</u> type 1
-Pump failure → -Reduced COP	- ↓ plasma volume → -decreased perfusion	1-Endothelial damage → <u>DIC</u> 2.Leukocyte – mediated → V.D.	Reflex Hypotension	Vasodilatation & increased permeability

Post mortem picture of Shock

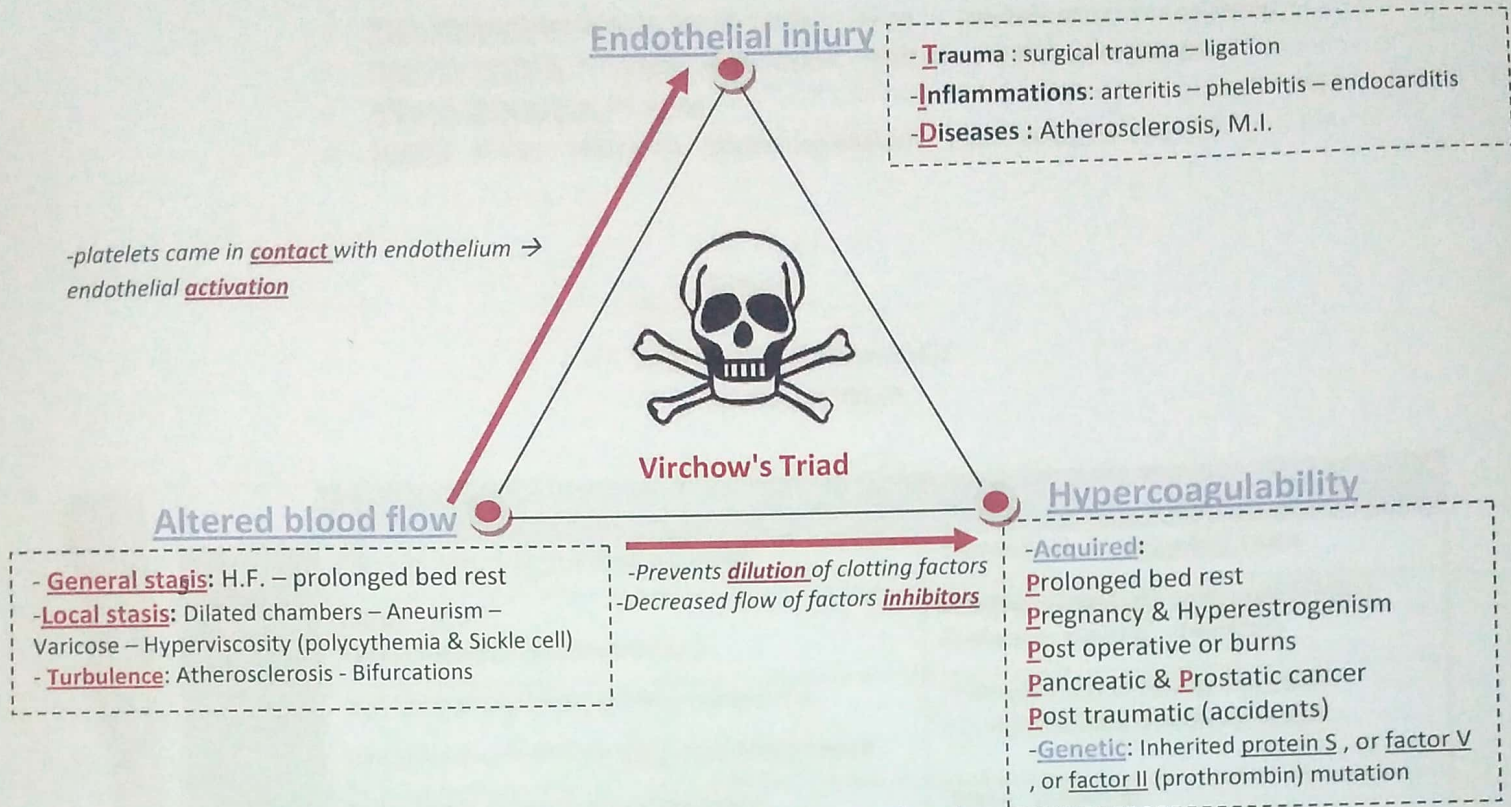
- Adrenal cortex → depletion
- Vital organs → degeneration & necrosis
- Vasodilatation & increased permeability in organs → Organ hemorrhage , & edema



Thrombosis

	Thrombus	Clot
Def.	<u>Solid</u> mass of blood elements, formed in <u>circulating blood</u> , inside CVS, during life	Coagulation of fibrin in <u>non- circulating blood</u>
Flow	<u>Circulating</u> blood stasis or turbulence	Non-circulating blood <u>stagnant</u>
Sites	<u>Inside CVS</u> during life	Outside CVS : wound clot Inside CVS: after death – on top of thrombus
Composition	<u>Platelets</u> + fibrin + blood cells Lines of <u>Zahn</u> present	No platelets No lines of Zahn
Attachment	adherent	Loose
Consistency	Firm or friable - Rough	Soft gelatinous - smooth

Causes of Thrombosis



Mechanisms of Thrombosis

Endothelial injury:

- **Adhesion of platelets**
 - Endothelial injury → Release of coagulation factor VIII (Von Willbrand)
 - Factor VIII acts as a bridge → adhesion of platelets to collagen of B.V.
 - Platelets are arranged in ridges (lines of Zahn): homogenous basophilic
- **Activation of intrinsic pathway** → activation of Fibrinogen → fibrin
 - Fibrin deposition between lines of Zahn
 - RBCs & WBCs may be seen trapped within fibrin
- **Release of Tissue factor** → extrinsic pathway → excess fibrin deposition
- **Depletion** of Prostacyclin (anti platelet) & Plasminogen activator (fibrinolytic)

- **In Cancers** → secretion of procoagulants → hypercoagulability
- **In pregnancy & contraceptive pills** → excess estrogen → Liver secrete → coagulation factors & decreases Prothrombin III → Hypercoagulability
- **Old age** → decreased PGI₂, increased platelet aggregation
- **In SLE** → antiphospholipid → decreased PGI₂, increased platelet aggregation + decreased protein C

Morphology of thrombus

- **Pale thrombus** In Arteries or cardiac - Firm - (contain platelets and fibrin)
- **Red thrombus** In complete occlusion – Soft -(more RBCs-less platelets)
- **Mixed thrombus** In veins
- **Septic** Bulky - yellowish (containing bacteria, septic emboli → pyemia)

Sites

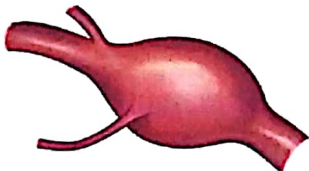
Venous Thrombosis

the most common



	Phelebo-Thrombosis	Thrombo-Phelebitis
Def	Thrombosis in <u>non</u> -inflamed veins	Thrombosis in <u>inflamed</u> veins
Causes	Stasis or H.F. Hypercoagulability (enumerate.)	Infection (Septic thrombo-phelebitis) Radiation ,trauma, chemicals
Sites	-Superficial (saphenous) with varicose vein -Deep Venous Thromosis DVT (calf, ileofemoral)	Inflamed veins of nearby infection e.g. Appendicitis, puerperal sepsis
Fate	-Superficial → Thrombus propagation -DVT → Pulmonary Embolism	-Septic → Pyemia

Migratory Thrombophlebitis: Recurrent thrombosis in different veins in case of cancer Pancreas (Trousseau's syndrome)



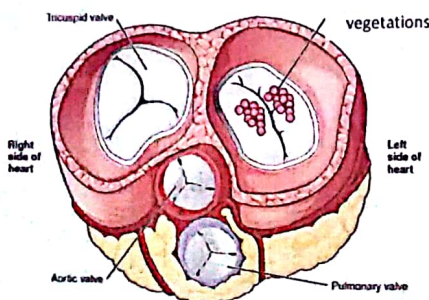
Arterial Thrombosis

Arteritis, e.g. PAN– Aneurism – Atherosclerosis
common in coronaries , cerebral at site of bifurcations

Capillary Thrombosis

DIC - Vasculitis - acute inflammation

Cardiac Thrombosis



1. **Atrial Ball -valve:** in atrial dilatation with M.S.
2. **Atrial Mural:** attached to endocardial surface
3. **Vegetations** (on cardiac valves): diseased valve e.g Rheumatic endocarditis, hypercoagulability
4. **Ventricular Mural:** On top of Myocardial infarction
5. **Agonal** inside right ventricle & pulmonary at the time of death

Fate and complications of Thrombosis

- Small recent thrombus → Dissolution (by fibrinolytic system)

N.B. fibrinolytics are useful in recent thrombi only. Old ones with extensive fibrin are resistant.

- Occluding or old thrombi:
 - **Organization** : heal by GT formation inside blood vessel → scar
 - **Canalization** : canal is open inside organized thrombus
 - **Calcification 'dystrophic'** : (phlebolith) detected by x-ray
 - **Embolization**: Septic → pyemia , Aseptic → ischemia, infarction
 - **Complication**:
 - Arterial : ischemia , infarction, gangrene
 - Venous: congestion , edema
 - **Propagation**: multiple clots on top of multiple thrombi → propagation → reach the right side of the heart

D.I.C.

Disseminated intravascular coagulation : widespread thrombi in multiple vessels with associated bleeding tendency

Causes:

- Obstetric complications (e.g. missed abortion)
- Cancers
- Septicemia

Pathogenesis:

Activated coagulation system → multiple thrombi in blood → consumption of platelets & fibrinogen → Bleeding tendency with activation of fibrinolytic system

Complications:

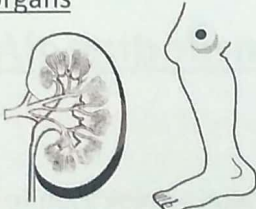
- Ischemic infarctions in multiple organs (Brain – Renal – Lung)
- Fatal Bleeding



Embolism

Embolus is a circulating, insoluble solid, liquid or gas. Embolism is impaction of embolus

Thrombo-Embolism (Detached thrombus)

	Pulmonary Embolism	Arterial embolism	Paradoxical embolism	Septic
Origin Of thrombus	Venous -Systemic <u>veins</u> (DVT) -Rt. side of the heart	Arterial - <u>Arteries</u> (3 A ?) -Cardiac from left side (vegetations, Mural)	Venous Systemic <u>veins</u>	Venous Infamed <u>Veins</u> Septic thrombophlebitis
Fate	Embolism of <u>pulmonary</u> artery or its braches 	Embolism of arteries supplying <u>limbs</u> or <u>organs</u> 	Embolism of arteries supplying <u>limbs</u> or <u>organs</u> (?) <i>(embolus reach right side of the heart then pass to left side through ASD, VSD)</i>	Lung or Liver 
Effect	- very Small emboli → organization (fibrous web) - Small size → 1-lung <u>infarction</u> (if the lung is congested) 2-or ruptured branch → <u>hemorrhage</u> 3-Or multiple emboli → <u>pulmonary hypertension</u> & Rt. S. H.F. - Large Saddle embolus → occlude >60% of pulmonary trunk → Acute <u>heart failure</u> → Sudden death in minutes <i>Death due to heart failure & serotonin in thrombus</i>	(if the collaterals are not sufficient) - Organ ischemia → infarction - Limb ischemia → gangrene	(if the collaterals are not sufficient) - Organ ischemia → infarction → gangrene - Limb ischemia → pale, pulsless, and cold	Portal Pyemia Pulmonary pyemia Systemic pyemia

Fat embolism

Aetiology:

- Fracture of long bone
- Severe trauma or burns

Pathogenesis: shortly within 1- 3 days in 1% of cases:

- Fat globules → emboli in veins → reach pulmonary → mechanical obstruction → **Dyspnea**
- Brain → irritability B.M. → depression
- 10% → Fatal

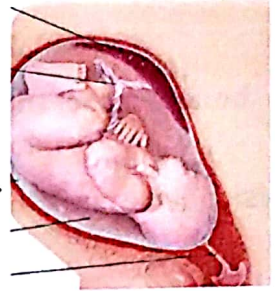
Amniotic fluid embolism

Aetiology:

- Vigorous uterine contractions during labor. In 1/50000 delivery

Pathogenesis:

- Amniotic emboli in injured uterine veins → reach pulmonary artery → mediators → **Pulmonary spasm** → **Acute right sided H. F.** (fatal)
- Chemicals in Amniotic fluid → **DIC** → severe bleeding



Air embolism

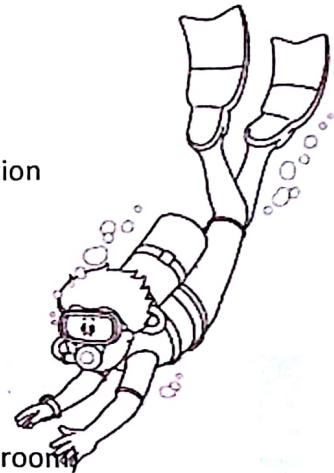
Aetiology:

- Accidental: Injury of neck veins – injury chest veins – tubal insufflation
- Decompression sickness (caisson disease)

>100cc → large bubbles → right ventricular failure

Pathogenesis of Caisson disease:

- During deep diving → high pressure → nitrogen gas dissolve in blood
- Sudden release of pressure → gas is forming bubbles in blood
- Gas bubbles → Dyspnea, muscle and joint ischemia (bends)
(treated by forced pressure, then gradual decompression in specialized room)



Tumor emboli (metastasis)

Parasitic emboli

Shistosoma → periportal fibrosis

Hydatid → liver Cyst

Ameoba → Amoebic Liver Absces

Ischemia

Decreased arterial blood supply

	Acute Sudden/ complete occlusion	Chronic Gradual/ partial occlusion
Causes	-Outside: <u>Ligation</u> , twisting, compression -Vessel wall: <u>Complicated atheroma</u> (hemorrhage) – spasm (Reynaud's disease) -Lumen: <u>Thrombus</u> – embolus	<u>Atherosclerosis</u> <u>Arteritis</u> <u>Arteriosclerosis</u> (in hypertension)
effect	Infarction In case of : -Organs with <u>end arteries</u> (retina, coronary, spleen, mesenteric, cerebral, renal) -<u>Rapid</u> occlusion -Tissue with <u>low vulnerability to hypoxia</u> (brain: 3 min , Heart: 30 min) -General Hypoxia (anemia, H.F.)	<i>If collaterals are insufficient</i> Atrophy & fibrosis Pain & claudications (accumulated metabolites) Infarction (in case of severe hypoxia)

Infarction

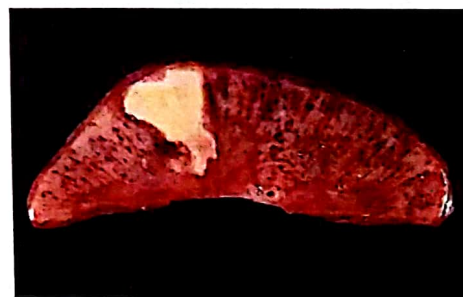
Coagulative necrosis of tissue due to arterial or venous occlusion. Liquifactive necrosis in CNS.

Causes of infarction: causes of acute ischemia (*enumerate*)

Gross picture of infarction:

Aseptic

- **Shape :** wedge, pyramidal (*Distal base, proximal apex*)
- **Surface(base):** Bulging then → Retraction (*later fibrosis*)
- **Color:**
 - Pale (*in solid organs : Heart, spleen , kidney*)
 - Red (*Intestine, Lung, gonads*) because of dual blood supply, Loose tissue, & congestion just before necrosis
- **Borders:** Hyperemic (Acute inflammation)
- **Covering serous sac :** Thick opaque (Fibrinous inflammation)



Septic (caused by septic emboli – or infection of infarction)

- Yellowish foci – surrounded by zone of congestion

Microscopic picture of infarction:

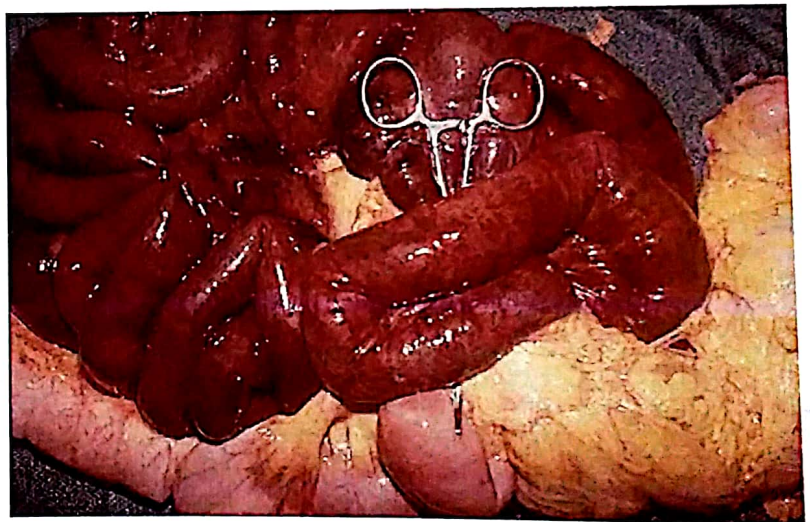
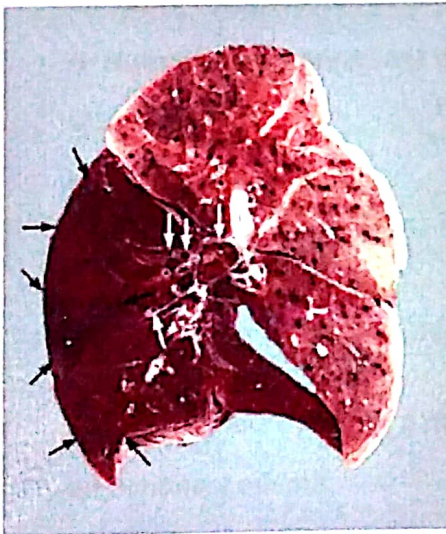
- Picture of **Coagulative necrosis** (*describe?*) → surrounded by inflammation
- In Brain infarction → microglia filled with fat (granular corpuscle)

Fate of infarction: -Healed infarction → fibrosis (gliosis in brain).

Clinical effects:

- Fever, Leukocytosis, elevated ESR (?)
- Elevated serum enzymes : Transaminases , CK (In M.I.)
- Cerebral: damage of pyramidal tract → hemiplegia
- M.I. : Arrhythmia → cardiogenic shock , Fibrosis → H.F.
- Lung: Hemoptysis (due to congestion) – Chest pain (due to pleurisy)
- Intestinal: Obstruction – gangrene & toxemia
- Renal: Hematuria – Senile atherosclerotic kidney (repeated infarctions)

Lung & intestinal infarction (red)



MCQ

1-All the following are possible causes of osmotic edema EXCEPT:

- a)Liver failure
- b)Nephritic syndrome
- c)Hypoproteinemia
- d)Protein-losing enteropathies

2-Lymphatic edema is:

- a)Generalized
- b)Induced by Thrombosis
- c)Non pitting
- d)Always reversible

3-All the following mechanisms are included in cardiac edema EXCEPT:

- a) Salt and water retention
- b)Increased hydrostatic pressure
- c) Increased permeability
- d)Decreased osmotic pressure

4-All the following causes may lead to Generalized edema EXCEPT:

- a)Left sided heart failure
- b)Right sided heart failure
- c) Severe proteinurea
- d) Radical mastectomy with axillary dissection

5- Congestion is:

- a)caused by arterial dilatation
- b)Caused by atherosclerosis
- c)Passive accumulation of blood
- d)All of the above

6- Nutmeg liver appearance is due to:

- a)Liver cirrhosis
- b)Fatty changes & congestion
- c)Hemosiderin & fibrosis
- d)All of the above

7- In Splenic congestion:

- a)Red pulp is atrophic
- b)White pulp is proliferated
- c)White pulp is atrophic
- d)All of the above

8- "Brown induration" is a result of:

- a)Pulmonary edema
- b)right sided heart failure
- c)Left sided heart failure
- d)none of the above

9- Which of the following diseases may lead to Hematuria?

- a)Malignant hypertension
- b)Vitamin K deficiency
- c)Thrombocytopenia
- d)all of the above

10- All of the following diseases presents with melena EXCEPT:

- a)Bleeding peptic ulcer
- b)Piles
- c)Ruptured varices
- d) both a&c

11- All of the following signs are seen in septic shock EXCEPT:

- a) Tachycardia
- b) Tachypnea
- c) Cold & wet skin
- d) Low blood pressure

12- Which postmortem finding is seen after shock:

- a) Adrenal depletion
- b) Cerebral edema
- c) Visceral hemorrhage
- d) All of the above

13- All the following features are found in Clot EXCEPT:

- a) lines of Zahn
- b) Devoid of platelets
- c) Blood is stagnant
- d) Found outside CVS

14- Which of the following situations lead to Thrombus formation:

- a) Polycythemia
- b) Portal hypertension
- c) Heart failure
- d) All of the above

15- Which factor is essential for formation of Irreversible thrombus:

- a) Factor VIII
- b) Thromboplastin
- c) ADP & Thromboxan
- d) none of the above

16- The number of propagating thrombi could be anatomically calculated as follow:

- a) Number of tributaries
- b) Number of related arteries
- c) Number of tributaries + 1
- d) Number of tributaries - 1

17- Features of arterial thrombus:

- a) Caused by prolonged bed rest
- b) Always red
- c) Grows against direction of flow
- d) all of the above

18- Agonal thrombus is formed inside:

- a) veins during death
- b) Left ventricle
- c) right ventricle and pulmonary
- d) systemic arteries

19- Features of DIC:

- a) Bleeding tendency
- b) Ischemic infarctions
- c) Platelet & fibrinogen deficiency
- d) All of the above

20- Thrombus which was formed in systemic veins and ends by cerebral infarction:

- a) Pulmonary embolism
- b) Thrombophlebitis
- c) Paradoxical embolism
- d) Atherosclerotic plaque

21- After a period of hospitalization following a car accident, the patient noticed swollen & tender calf. Short after, a sudden chest pain & dyspnea develops:

- a) Fat embolism
- b) Thromboembolism
- c) Arterial thrombosis
- d) Thrombophlebitis

22- Amniotic fluid emboli lead to:

- a) Pulmonary Vasospasm
- b) Massive uterine bleeding
- c) Chest pain
- d) All of the above

23- Caisson's disease is an example of:

- a) Pulmonary thromboembolism
- b) Systemic Thromboembolism
- c) Fat embolism
- d) none of the above

24- Red infarction is seen in:

- a) Heart
- b) Kidney
- c) Lung
- d) none of the above

25- Examples of moist gangrene include all of the following EXCEPT:

- a) Gangrene following Strangulated hernia
- b) Diabetic gangrene
- c) Crushing limb injury
- d) Atherosclerotic limb

26- All the following features are found in Moist gangrene of limb EXCEPT :

- a) Putrefaction
- b) Line of separation
- c) Toxemia
- d) Ischemia

27- Reynaud disease is characterized by:

- a) Arterial vasospasm
- b) aggravated by cold
- c) Cyanosis of extremities
- d) all of the above

28- Gas gangrene of the deep wound occurs in the presence of:

- a) Arterial ischemia
- b) Venous occlusion
- c) Anaerobic Clostridia
- d) Aerobic bacteria

29- Black color of gangrene is due to:

- a) Hemosiderin
- b) H_2S
- c) Iron Sulphide
- d) Tissue necrosis

30- Generalized edema results from all of the following, EXCEPT:

- a) Systemic hypertension
- b) Congestive heart failure
- c) Cirrhosis
- d) Hyperaldosteronism

31- Embolism occurs in:

- a) Bilharziasis
- b) Leukemia
- c) Bone fracture
- d) All of the above

32- All are characteristic of dry gangrene EXCEPT:

- a) Slow progression
- b) Marked toxemia
- c) Black discoloration
- d) Self separation may occur

33- In Chronic venous congestion of the lung, alveoli show all ,EXCEPT:

- a) Heart failure cells
- b) RBCs
- c) Transudate
- d) Exudate

34- Thrombosis is more common in:

- a) Veins
- b) Arteries
- c) Cardiac valves
- d) Left atrium

35- A thrombus over myocardial infarction is called:

- a) Ball thrombus
- b) Mural thrombus
- c) Vegetation
- d) none of the above

36- The following are factors enhancing Venous thrombosis EXCEPT:

- a) Endothelial damage
- b) Stasis
- c) Increased fibrinogen
- d) Decreased plasma proteins

37- The effect of embolus depends on:

- a) Collaterals
- b) Infection of embolus
- c) Size of embolus
- d) All of the above

38- Which type of shock is associated with massive pulmonary embolism:

- a) Hypovolemic
- b) Cardiogenic
- c) Neurogenic
- d) none of the above

39- All the following leads to arterial thrombosis EXCEPT:

- a) Thrombangitis obliterans (Burger)
- b) Poly arteritis nodosa
- c) Atherosclerosis
- d) Heart failure

40- The most common origin of pulmonary emboli is:

- a) DVT
- b) Varicose veins
- c) Cardiac diseases
- d) Valve diseases